REMARKS

Claim 9 is pending in the instant application. Claim 9 has been rejected. Claim 9 has been amended. No new matter has been added by this amendment. Reconsideration is respectfully requested in light of the following remarks.

I. Rejection of Claims Under 35 U.S.C. §103

Claim 9 remains rejected under 35 U.S.C. §103(a) as being unpatentable over Moyer et al. ((Aug. 1999) *Am. J. Physiol.* 277(2 Pt 2):F271-6) in view of Cormack et al. ((1996) *Gene* 173:33-38) further in view of Chou et al. (1991) *J. Biol. Chem.* 266:24471-24476) for the reasons of record. In response to Applicants arguments that the cited references do not teach a mutant human CFTR, the Examiner suggests that because the CFTR of Moyer et al. was manipulated to create the CFTR-GFP construct, the CFTR is a mutant. Applicants respectfully disagree with this rejection.

It is quite clear from Applicants' disclosure that a mutant CFTR is intended to include ΔF508 CFTR and CF mutation in this functional class. See page 6, lines 3-22. However, in an earnest effort to facilitate the prosecution of this application, Applicants have amended claim 9, as supported by the disclosure at page 6 (lines 21-22), to indicate that the CFTR mutant is ΔF508 CFTR. As indicated in Applicants' response filed August 8, 2005, and reiterated herein, Moyer et al. do not teach or suggest the use of a nucleic acid construct encoding ΔF508 CFTR-GFP in the method disclosed therein. This reference teaches that butyrate is useful for treating CF and focuses on the effect of butyrate on renal

function as CF patients exhibit renal dysfunction. See the paragraph bridging column 1 and 2 of page F271. Moyer et al. disclose the use of a GFP-CFTR construct to monitor CFTR expression and localization in renal cells exposed to butyrate because expression of endogenous CFTR in renal epithelia is low and difficult to detect or below the limit of detection. See page F271, column 2, lines 26-31. However, there is no teaching or suggestion to modify the teachings of Moyer et al. to employ a nucleic acid construct encoding ΔF508 CFTR-GFP to identify agents which increase functional cell surface expression of a ΔF508 mutant CFTR protein. In so far as Cormack et al. teach mutant GFP and Chou et al. teach transcriptional regulatory elements of CFTR, these references fail to overcome the deficiencies in the teachings of Moyer et al.

Because Moyer et al., Cormack et al. and Chou et al. fail to teach or suggest the use of a ΔF508 mutant human CFTR cDNA coding region and a cDNA of an EGFP reporter gene linked at the 5′ end to the mutant human CFTR cDNA coding region and wherein said cDNAs are under the regulation of the proximal human CFTR promoter region these references cannot be held to make the present invention obvious. It is therefore respectfully requested that this rejection be reconsidered and withdrawn.

II. Conclusion

Applicant believes that the foregoing comprises a full and complete response to the Office Action of record. Accordingly,

favorable reconsideration and subsequent allowance of the pending claims is earnestly solicited.

Respectfully submitted,

Jane Massey Licata
Registration No. 32,257

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Licata & Tyrrell P.C.
66 E. Main Street
Marlton, New Jersey 08053

(856) 810-1515